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ANOREXIA NERVOSA

Malvin Cole, M.D.,* Belinda Straight, M.D.,† Mary Robinson, M.A.,‡ Reginald S. Lourie, M.D.§

INTRODUCTION

Dr. Lourie:

From the way anorexia nervosa as a symptom looks to the outsider I think we might give it a new name: the "mule-bear" syndrome. The mule part refers to the stubbornness of these patients in relation to the food intake which is the most frustrating kind of symptom. The bear part refers to the relative physiological hibernation. In this pattern of self-imposed starvation, the human body goes into a form of hibernation in an attempt to maintain the homeostatic patterns that will keep life going in spite of inadequate nutrition.

CASE REPORT

This was the first Children's Hospital admission of a 13 year old white adolescent girl who was entered because of a weight loss of 40 pounds in six months.

Prior to July 1956 the patient was described as a normal, active, intelligent girl who did good school work, liked dramatics, weighed 110 pounds, and had had regular menses for six months. Her father had died in April 1956 of heart disease, and her mother had then taken full time employment for the first time. The girl had shown minimal grief at her father's death.

In July 1956 she began to diet in order to reduce her weight and especially her waist line. At first her mother accepted this dieting as a temporary fad, but by September 1956 was genuinely concerned. Supposedly, the girl had been teased about her weight while at camp. By fall of 1956 both mother and family physician began to advise against this diet. The girl would listen to such advice only to refuse openly at home to eat adequately. She would alternate between several days with no apparent food intake and a single excessive meal followed by crying and complaint of gas and abdominal discomfort. She continued to lose weight rapidly. She professed a desire to be "pretty" and wanted a smaller waist. She became increasingly interested in religion, desired to sit alone in a corner of her classroom, would spend much time alone in her room at home behind a closed door, and was often hostile and irritable to her mother. Shortly before admission her school performance had markedly changed to failing grades. She was seen by a psychiatric consultant prior to admission to this hospital and testing was done. It was noted both at school and at the psychiatrist's office that the daughter showed aversion to physical contact with the mother.

In January 1957 the patient was admitted to Children's Hospital Adolescent Unit.

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She now weighed 70 pounds, had had no menses for six months, noted increased sensitivity to cold, decreased perspiration, increased constipation, decreased breast tissue, and increased dryness and coarseness of her skin. She denied headaches or neurologic symptoms. She had no vomiting and no anorexia. She avoided all foods because when she did eat, she would eat excessively; this resulted in belching, expelling of flatus, and abdominal discomfort. She had no abdominal pain.

Her previous growth and development had been normal. Her past medical history was non-contributory. Her mother had been diagnosed as a pseudo-hemophiliac and her father had died of heart disease. She had no siblings.

When admitted to the hospital the patient had a temperature of 97 degrees, a pulse rate of 48, blood pressure of 80/60 and a weight of 69 pounds. She was an emaciated white girl looking chronically ill and older than her stated age, with slender extremities and trunk, drawn facies, and bright alert eyes. She was hyperactive and emotionally labile. Her skin was dry and scaly and there was marked generalized loss of subcutaneous tissue. Her teeth were in good condition. There was a pronounced bradycardia. Her abdomen was scaphoid but soft, and no masses were palpable. She had a normal amount of pubic hair and moderate breast development. Her hands and feet were cold and mildly cyanotic. Complete neurologic examination was normal.

Laboratory studies revealed a hemoglobin of 13 gm. per 100 ml., hematocrit 40 per cent, and white blood cell count of 8300 with a normal differential. Her urine concentrated to a specific gravity of 1.032 and was otherwise normal. An intradermal tuberculin test was negative. X-ray examination of the skull revealed a normal sella turcica with no abnormal calcifications and no evidence of increased intracranial pressure. Her bone age was normal. X-ray of the chest revealed a small "teardrop" heart and a relatively enlarged liver. X-ray examination of the upper gastrointestinal tract after ingestion of barium sulfate showed prominent gastric rugae and complete gastric emptying in two hours. An electroencephalogram was reported as abnormal with no focus, and a generalized low amplitude.

Blood cholesterol was 72 mg. per 100 ml. A total eosinophile count was 50 per cu. mm. prior to ACTH injection but 0 per cu. mm. after ACTH injection. Urinary 17 ketosteroids were 0.97 mg. per 24 hour specimen prior to ACTH therapy, and 1.85 mg. per 24 hours on the day following ACTH stimulus. Basal metabolic rate determinations on two occasions at two separate hospitals were minus 40 per cent and minus 61 per cent. A serum protein bound iodine was 10 mcg. per 100 ml.

After one month of medical and psychiatric study the patient was transferred to a psychiatric unit for further therapy. At first she was difficult to manage and lost weight to 67 pounds. She poured food down the toilet and lied about the amount that she consumed. She cried when an attendant was placed in the room at meal time. She exhibited great energy and physical activity including exercises when alone. She was greatly concerned by the food intake of other patients and showed much interest in feeding others, taking food orders, and carrying food trays.

Her therapy included psychiatric interviews, and later the addition of a tranquilizer, meprobamate, 100 mg. three times daily. Before discharge she weighed 71 pounds, and between nibbling from other trays and eating sporadically from her own, had an improved caloric intake.

DISCUSSION

Dr. Cole:

In discussing this case we might begin by going over some of the points in the history that are typical or atypical, and then discuss points for which

to look in future cases. The first fact to note is that this is a 13 year old girl. This disease is usually found in females in a ratio of about 4 to 1 over males[1]. The commonest age incidence is from about 16 to 28 years with some slight variation in different series. In males the disease usually occurs in a slightly older age group; it may occur in people over 40 years of age and in post-menopausal women. This latter group comprises about five per cent of the total tabulated cases in the literature. Most patients with this illness are unmarried.

The second fact to note is that our patient had her menarche six months prior to the onset of the illness or at about the normal age. Usually menarche has been reported to occur late, often not until 19 years of age. These patients are characterized as being small in stature and late in maturing sexually.

I cannot say how often this condition will occur in Negroes, if it does at all, because most of the literature until the last few years originated in England where one sees very few negro patients.

This girl's father died three months prior to the onset of the illness. The syndrome has often been noted to follow such events as the death of a parent, homesickness, or impending marriage. It can also follow some combination of physical and emotional illness or trauma, and I use the term "combination" because it does not seem to be a mechanical effect of trauma so much as a psychological reaction to trauma. One wonders if the syndrome of anorexia nervosa would have been averted in this girl if a normal amount of grief had been shown.

There has been some neurophysiological investigation on what kind of experimental lesions can cause loss of appetite, or bulimia. Such lesions have usually been localized in the hypothalamus and the frontal lobes (especially their orbital surfaces or premotor areas). Lobotomy has caused disturbances in gastrointestinal physiology as well as the appetite mechanism. Some organic neurological diseases have been known to precede the onset of anorexia nervosa; these include encephalitis, Parkinsonism, narcolepsy, usually of the secondary type, and pituitary disease.

This case began with voluntary dieting in the face of a history of preexisting obesity. Since the time of Gull this has been noted to be a characteristic finding[2, 3]. These people are often teased about their obesity as indeed this girl was. Often the disease begins with some voluntary attempt at weight reduction which seems to get out of control. The two children reported in detail by Falstein, Feinstein and Judas[4] were both obese and both began to diet in imitation of their mother who was dieting. The pattern of limiting their food intake then became fixed.

These people are also often found to be in the food industry and have a fixation on food, as it were. In the history one might ask how important food is in the family and in the family life. It is usually quite important

and one usually finds that the mother has stressed it to a large degree. One other fact that might be ascertained in the history is how the mother reacts to small or large emotional trauma. It has often been noted that the mothers will react with minimal or more severe anorexia themselves, thus setting the pattern for the child.

This girl, as in so many of the other cases reported, had alternating periods of bulimia; she would often stuff herself and then not be able to eat for a few days. This brings up the question of whether anorexia really exists in the syndrome. Many authorities have doubted whether there is really a loss of appetite, and some have even claimed "that there is an obsession with food and a compulsion not to eat"[5]. This girl also complained of some abdominal discomfort which is a complaint often preceding the onset of anorexia nervosa. Often the patients will give a history of having severe abdominal discomfort or pain, and stop eating to try to control this. Eventually they become fixed in this pattern. The gastric spasm and pain can, on the other hand, follow the onset of the syndrome. The relationship of gastric spasm to psychophysiological mechanisms does not have to be elaborated; we all know it is considerable.

Our patient also claimed that she wanted to be pretty, and wanted a small waist. This could mean that food in general has been related to acceptance or rejection by her peers or by her parents. It may also be a denial of sexual or social maturation.

Our patient had amenorrhea for about six months. Many authorities have claimed that amenorrhea must be present for the syndrome to be diagnosed. Of course this would immediately eliminate the diagnosis in males. Other authorities such as Berkman, in 1930, reported amenorrhea to be present in 53 per cent of the cases[6]. Amenorrhea may occur prior to the anorexia itself, it may come on about the same time as the anorexia, or it may follow the anorexia by some months. Thus the time interval between the anorexia and the amenorrhea is not exactly clear. Again this might be some denial of sexuality, but the English usually consider it either a primary finding, a manifestation of secondary hypopituitarism, or simply the effects of inanition.

Our patient was also constipated, but had no history of vomiting, so that the basic syndrome of refusal to eat, amenorrhea, constipation, and vomiting was not complete. Vomiting has been found in about 56 per cent of cases in large series, among which Berkman's was one of the largest[6]. When present, vomiting usually occurs after eating, especially when food is forced. It has also been attributed to gastrointestinal spasms. Besides being important in the diagnosis of the disease, vomiting is also a major problem in management because it increases both the existing malnutrition and the electrolyte imbalance that is often present. Because of the vomiting

it is frequently very difficult to evaluate exactly how much food these patients are taking in. Often they will lock themselves in the bathroom to vomit. One of the reported cases vomited into his toy chest which he then hid[4]. It has been noted that in the early stages many of these patients will stick their fingers down their throats and thus cause vomiting. This may become uncontrollable after a few days and the patient vomits whether he wants to or not. It is important to note that these patients quite often object to being watched, even when this is done simply to find out exactly what their food intake is.

Other complaints occur less commonly. Hematemesis has been reported. Decreased thirst has also been reported, but in a rare case increased thirst was noted to such a degree that a diagnosis of diabetes insipidus was entertained. Lastly, abdominal pain usually in the epigastric or right lower quadrant has been reported. Some of these patients have even been operated on for appendicitis.

The vital physical signs in this case are quite characteristic. Her temperature, pulse, and blood pressure are decreased. As far back as the time of Gull it was noticed that this was characteristically the case. That this is specifically related to anorexia nervosa as a dynamic mechanism is doubtful. It is probably a homeostatic mechanism to maintain life at a lower level of metabolism. It was also noted that there was a 40 pound weight loss. This is important for gauging the prognosis of the disease since most of these patients are admitted to the hospital with some degree of weight loss. Dejerine has claimed that any patient who loses one-half of her body weight has a very grave prognosis, and, in fact, some of the more severe cases were termed by Ryle and others as anorexia psychotica rather than anorexia nervosa[7]. This patient has lost 40 per cent of her weight and it would probably be well to keep her from losing too much more. Another factor which makes the prognosis worse is a recurrence of symptoms in someone who was thought to have once recovered from this condition. (One of the factors contributing to a grave prognosis is the great degree of secondary gain that these patients often get from this syndrome.)

In recent years it has been claimed that between 75 and 90 per cent of these patients will recover, most of the later series tending toward the higher figure. It has been claimed that amenorrhea is usually the last symptom to disappear and perhaps the best criterion of cure.

Everyone who has seen this patient has noted her intense hyperactivity. Every time I passed her room she was running out into the hall and literally tearing around the corners so that no one could speak to her casually. This has also been commonly noted and is quite characteristic; until the last stages of cachexia these patients are hyperkinetic[1].

Her skin was characteristically noted to be dry, scaly, cold, and cyanotic.

This probably is in conjunction with the decrease in her metabolism. Vitamin deficiencies have been seen much more rarely than one might expect with such severe cases of malnutrition. One might look for some pigmentation, usually described as brown, and usually found over the abdomen. This also raises in the examiner's mind the differential diagnosis of such things as Addison's disease. In the very severe cases contractures have been reported, as have decubitus ulcers.

Our patient was noted to have atrophy of her breast tissue. Characteristically this is not found, in contrast to hypopituitarism where it is commonly seen. Even in the case of severe cachexia, breast tissue is usually preserved. The only explanation that one might have for the breast atrophy present in this case is the short period between the onset of menarche and the onset of the syndrome.

This patient, as is characteristic, showed "la belle indifference" toward her symptoms. These patients, although anxious in a sense, do not show their anxiety. When this girl was asked why she came to the hospital she answered "just for some tests because I am a little thin". She was completely apathetic toward the severity or the ugliness of her own symptoms.

This is one disease where blue sclerae have been noted. Peripheral venous thrombosis of the lower extremities has been seen occasionally and is usually attributed to dehydration. Some of the more severely affected cases have edema which is usually attributed to hypo-albuminemia. Delirium and severe scaphoid abdomen may result, and these patients are so cachectic that they resemble patients with severe esophageal carcinoma. (Osler[12]).

The girl's blood count was normal although these patients usually show a high degree of anemia, the exact type of which is not mentioned in the literature. Her heart was described as a "tear-drop heart" of small dimensions. This microsplanchnia has been attributed by some investigators of metabolic disorders to a secondary homeostatic mechanism. The low BMR, usually in the range of minus 20 to minus 40 per cent, is quite characteristic, and has also been attributed to inanition. Berkman, however, denied that inanition itself can ever lower the BMR, and called this an independent psychophysiological mechanism[6]. It has been considered by some authors to be a manifestation of secondary hypopituitarism.

There are other findings in this case that might not fit in the diagnosis and they should be mentioned. The first is a serum protein bound iodine of 10 mcg. per 100 ml.; another is the abnormal electroencephalogram, and the third is the relatively large liver. These people frequently have a pluriglandular deficiency, usually secondary to the disease itself, although before this syndrome was separated from organic hypopituitarism the two conditions were considered to be identical. It would be quite

inconsistent, therefore, in the face of a supposed glandular deficiency for a serum protein bound iodine to be even at a high normal level. The abnormal electroencephalogram bears further investigation; even though this seems to be a classical case of anorexia nervosa, the possibility of intra-cranial pathology should be considered[8]. The enlarged liver remains to be explained. It might possibly be due to the marked cachexia enabling the liver to be more easily palpable. It could perhaps be due to some hepatic pathology, i.e. nutritional liver disease.

Other laboratory abnormalities which have been characteristically noted in previous cases include a decreased gastric acidity (in some cases, gastric anacidity), and a flat, slow rising glucose tolerance curve. The slow, flat glucose curve has been attributed to 1) poor absorption of glucose by the intestinal tract, plus rapid utilization, 2) inability to phosphorylate absorbed glucose due to secondary adrenal hypofunction, and 3) secondary hypopituitarism.

The most important disease to rule out in the differential diagnosis of anorexia nervosa is Simmond's disease, or panhypopituitarism, which usually occurs in a female to male ratio of about 2 to 1, whereas in anorexia nervosa the ratio is about 4 to 1. Young people under 20 would be more likely to have anorexia nervosa, while Simmond's disease is characteristically seen in older people. In addition, people who have anorexia nervosa usually have not been married; of those who have, most have no children. Contra-wise, the commonest cause of Simmond's disease is the Sheehan-Murdock syndrome of postpartum hemorrhage with pituitary necrosis.

Other causes of hypopituitarism and Simmond's disease must also be ruled out. The commonest cause in this age group would be a tumor in or about the pituitary fossa, and the tumors which should first be thought of are craniopharyngioma and enlarging glioma of the optic chiasm. Other causes of hypopituitarism are trauma to the base of the skull, infection, granuloma, such as sarcoidosis, and also, quite rarely, leukemic infiltration of the pituitary gland.

Another point in the differential diagnosis between anorexia nervosa and Simmond's disease is weight loss. According to Dr. Lawrence Kyle[9], weight loss in Simmond's disease is a very late manifestation and is usually terminal, whereas weight loss in anorexia nervosa is an early manifestation. Also, people with Simmond's disease usually have decreased energy, whereas people with anorexia nervosa are hyperactive. Patients with Simmond's disease characteristically have poor teeth with multiple caries and poor dental hygiene, whereas the teeth are well preserved in anorexia nervosa. Anorexia nervosa is frequently characterized by hirsutism, usually a fine lanugo type of hair over the posterior shoulders. In contra-distinction,

people with Simmond's disease usually have a loss of hair from the pubic area, axillae, head and eyebrows. The breasts in a patient with anorexia nervosa are usually well preserved, while in Simmond's disease they show marked atrophy in the early stages. The incidence of amenorrhea in both diseases has been contested. Some investigators believe that it is more common in Simmond's disease; others think that it is more common in anorexia nervosa. Eosinophilia is found in Simmond's disease, as is spontaneous hypoglycemia[10]. These are not often seen in anorexia nervosa.

Other conditions to be considered in the differential diagnosis would include some psychiatric disorders. Almost all have been mentioned in the literature, starting with conversion reactions and including early schizophrenic reactions.

Other endocrine diseases can resemble anorexia nervosa. Certainly myxedema can, as can hypopituitarism, diabetes insipidus, and also Addison's disease. Because of the pallor of their skin these people have sometimes been misdiagnosed as having anemia rather than anorexia nervosa.

Complications of anorexia nervosa include bone fractures due to calcium resorption, scurvy, and rarely other vitamin deficiency diseases such as pellagra. Hertz and Means[11] have reported thyrotoxicosis as an aftermath or sequel to anorexia nervosa. They think that low food intake can stimulate thyrotoxicosis. Addisonian crises can also be superimposed, perhaps due to secondary pluriglandular failure.

In the therapy of anorexia nervosa many things have been used. Insulin, pituitary hormones implanted in the omentum, and thyroid hormone, have been tried at one time or another, with and without success. Certainly, if there is success it is only symptomatic, and therapy must be used with caution since such things as insulin and thyroid hormone can certainly increase weight loss.

The main medical problem in this condition, especially in severe cases, is the immediate control of malnutrition. Tube feeding is generally not advisable but is sometimes absolutely necessary in the terminal stages. The five points in the medical management of these cases which have been commonly agreed upon are: 1) Separate these people from their family; 2) Hospitalize them; 3) Put them to bed; 4) Provide carefully for body warmth; 5) Give good nursing care. Concerning any further management, the literature varies widely.

Dr. Straight:

I would like to add a little to the history given by the patient's mother who was a quiet, thin, conscientious, intelligent woman, concerned about her child. Warm feelings gave her discomfort and the expression of these feelings was difficult for her. Dr. Cole mentioned that there was a min-

imal reaction of grief in this household on the death of the father. Both the patient's and the mother's feelings of loss were held in tight control.

Both parents were intellectuals, rational, controlled and holding high standards of behavior for themselves and for their child, to which she was responsive. The father's early life was one of self discipline with emphasis on not showing one's inner feelings, and with a strongly idealistic trend. He went through college and married a young woman who had similar interests to his own, and whose personality makeup was somewhat like his. He became an engineer, volunteered for active duty in the Air Force although he was aware of a damaged heart, and was restless when he was turned down. He continued to work as an engineer until he was forced to stay at home because of his poor health.

The mother, also a college graduate, had been attracted to him for his qualities of control and intellectual interests, and had a rather close relationship with him, particularly during the last year of his life when he gradually became able to confide his doubts and fears to her, and when she then helped sustain him. Both mother and daughter were unable to describe the father, how he looked, what he was like.

The patient was a wanted baby. The delivery was a normal spontaneous one. The mother had asked for a caudal anesthesia so that she might be awake for all of the delivery. "She was a nice little baby, and we were glad to have her". She was breast fed for a few days then put on a bottle. Weaning was done at a year by "just putting away the bottle". She had been on a four hour schedule for feeding during the first months.

The mother did not recall any feeding difficulties prior to the present illness. Toilet training was started at a year and the patient was dry during the day at two years. There was no thumb sucking but the patient began to bite her nails this year.

There was a minimal amount of cuddling. It is not clear whether the cues for this came from mother or from child but I would think from the mother. There were times when the baby cried in the playpen and her mother wondered about picking her up, but often decided against it for fear of spoiling her.

The child attended nursery school and entered the first grade at age six. There were no troubles at separation from home to go to school, and school became a source of accomplishment and pleasure for her. Like many patients with anorexia nervosa she was a quick student and a good scholar until the onset of her illness when her grades declined. She entered many extra curricular activities, and had a few close friends of the same sex. Outside interests included church groups.

Her mother described the patient before she got ill as rather cheerful, conscientious, capable of having fun and having a few good friends.

Six months after her father's death the patient was teased at camp about having a slight bulge at the waistline. She actually had a fairly good figure then, having been plump at age 10. She resolved to make her stomach smaller. She cut down her food intake drastically without telling her mother. Since the mother left early in the morning to go to work the patient would tell her mother that she had eaten her breakfast. Evening meals were also skipped, the patient saying she had already had hers. She entered into the many subterfuges which the patient with anorexia nervosa becomes wrapped up in, such as pretending to take the food and then disposing of it down the sink. It is this kind of pattern, where the child expects to be found out or watched, which makes it inevitable that the mother becomes engaged in watching, finding out and admonishing, feeling angry and dismayed.

Finally food was refused openly. By this time she had lost quite a lot of weight. She exercised vigorously, pounding her abdomen and crying, "It's always on my mind. It will never go away. It will never digest. I will have it always". On the other hand these periods of starvation were interrupted by sprees of gorging on cake and candy in solitude, and these in turn were followed by remorse. She also was concerned about constipation, often not having a bowel movement for a week at a time.

Menstruation, which had been established six months before, ceased with the onset of the illness. She said of this "Maybe I will never menstruate again and never have children". Several months ago she began to wear underpants under her pajamas, saying, "It is safer".

During the father's illness much attention was paid to the diet and its content. The father suffered from anorexia during his last six months of life, and there was some unspoken concern about what he was eating and how much. When he died the patient worried about the loss for grandmother. Only once did she mention her own loss, adding quickly, "but let's not bring that into it". On the other hand during the last year of her father's life the patient was able to do things for him more. She was certainly aware that her parents were tense and uncertain about his health, although they tried to keep her from having these concerns.

A depressive trend developed in the patient. Sometimes she said, "I wish I were dead". "Everyone wants to see his Lord". At another time she said, "I'm ugly, I'm stupid, I'm no good".

Her relationship with her mother became increasingly difficult. She felt watched; she felt physical repulsion toward her mother, not wanting to be touched by her, or eat with her. Privacy of her room, important in the teenager, was now a matter for open hostility, but at times the daughter showed great dependency and clinging like that of a much younger child.

The patient's stay in the hospital resulted in difficult management

problems for the ward. She was hostile to particular members of the nursing staff and aides, especially those connected with the feeding process. With other members she was more friendly, and like many patients with anorexia nervosa she took an interest in what the other patients were eating, pressuring them to eat more, and reporting them when they ate poorly.

The plan for her treatment was to have her medical care under the supervision of the pediatric ward resident, to have her seen daily by a member of the Department of Psychiatry, to have her work with occupational therapy where she liked to help with the younger patients, and to have the nurses encourage her to help on the wards. At the same time it was necessary to draw limits rather strongly, particularly about staying on the ward. It was emphasized with her that her ward care and management would be kept quite separate from the psychiatric sessions. In the psychiatric interviews there was much negative feeling toward the therapist, with somewhat more friendly attitude toward the ward personnel. During the last week of her hospitalization she grew more relaxed on the ward and gained four pounds.

When interviewed she was a gaunt looking teenager with long brown hair and thick dark brows. The boney structure of her face thrust its way through her pale skin. There was little change in her facial expression even when tears ran down her cheeks. Her eyes were sunken. She looked away much of the time, but observed the psychiatrist frequently. Her movements were quick. She darted down the hall and out of sight, or suddenly and silently appeared near a group of people who were talking. She found difficulty being in an open space such as a hall and preferred the enclosed rooms. Her mood was depressed, she cried frequently, and this was often while saying "There is nothing wrong with me. I am only here for some tests. I don't see why people keep fussing at me". Denial was prominent. There were no hallucinations or ideas of reference. She often sat cross-legged with her feet tucked in under her body, squirming frequently, or jumped suddenly in the air. She constantly felt her abdomen through her clothes. When asked if her stomach was different from other peoples' she replied, "No, you just don't understand". She was guarded about all of her feelings, and guarded about giving any information about her self. When asked what movies she liked she wanted to know why this had been asked. There appeared to be a great sensitivity and vulnerability in human relationships with a need to withhold from herself and from others things which were pleasurable. We see in the guarded behavior the fear that she was abnormal.

My impression was that this patient in early adolescence showed evidence of pathology in the mother-child relationship. There appeared to be con-

siderable confusion around the areas of eating and child bearing, even if they were intellectually understood on the conscious level. This patient seems to have regressed into some of her much younger fantasies about carrying babies and where they come from. We would suspect the fantasies of oral impregnation. The death of the father seems to have been a precipitating factor. Cessation of menstruation, the feeling of something in her stomach of which she was ashamed may be well tied together into a pregnancy fear. Unmet oral needs are prominent. We see behind the starvation the desire and fear of overindulgence. There was projection "It's your fault, these interviews are what make me unhappy". Denial was a prominent defense. Guilt and self-destructive impulses played a prominent role. Tension in mother-child relationships is often reflected in eating, and it would seem that this is a potent weapon which is brandished between mother and child, the child reacting later to earlier tensions which were felt over feeding. The child later withholds gratification to her mother by refusing to eat and also punishes herself.

This patient's assets included a strong interest in relationships with others, the ability and need to maintain friends throughout her illness, and the encapsulation of the feeding difficulties so that in other areas she was able to maintain her interests. It was felt that this patient needed long term therapy, starting in a controlled environment, so she was transferred to a psychiatric hospital. After three months of hospitalization and psychotherapy the patient was discharged home, having gained weight, and was restarted in school. She asked for a continuation of therapy after leaving the hospital. She was doing well, eating a well balanced diet on her own impetus, but still having problems in family relationships on which she is working with her therapist.

Mrs. Robinson:

I saw this child on the ward in an attempt to facilitate our differential diagnosis. Because we knew that she was a suspicious, rather guarded child, I saw her on two occasions previous to the actual examination. The first time her reaction to me was one of open suspicion, but she was quite willing to state her feelings, and when I told her frankly who I was and why I was there she seemed to respond to this, and to appreciate the fact that I had not tried to deceive her.

I found at this time, however, that she would either refuse to respond, or give monosyllabic answers to relatively innocuous questions about where she went to school, what she liked, etc. Her facial expression and her appearance were unvaryingly depressed. On occasion, tears would suddenly pour down her face but it was impossible to relate these episodes of crying to the external situation. At one point when she began crying I asked her if she felt like talking about what was upsetting her.

She answered, "I can't talk about it because I don't know what it is". By the end of the first interview she related to me to the extent that she was able to ask me to do a favor for her and arrange for friends her age to visit her on the ward. This, fortunately I was able to do.

At the time of the second visit she welcomed me with some small degree of warmth but made very sure that our contact was through the medium of a game. In this game she showed a great fund of general information and gave the impression of being a rather intelligent child. She was relatively relaxed until she heard the food carts being brought on the ward. Then she jumped out of bed, said "You'll have to excuse me now", and ran to the bathroom. Thus our interview terminated.

At the time of the examination I gave her a choice of coming to my office or remaining on the ward, and she quite enthusiastically said she would like to come to my office. I gave her the Rorschach test to which she was quite willing to respond, even though she commented that she had taken this test before. I gave her other projective materials, however, which she found quite threatening. She was either unable to respond, responded very superficially, or took an extremely long time to respond, moving about in the chair, biting her nails, turning her face away from me, and all in all presenting the picture of an extremely anxious child.

One point that was interesting was that several days after this interview the child accosted me in the hall. I had not seen her and she made a great point of calling to me. At this point she seemed to want to talk about something and was very eager for physical closeness, but this was the only occasion on which I got this impression. I think this shows some of the child's ambivalence about wanting to be close to people, yet finding it quite threatening to be close.

My over-all impression of her was that she was an extremely anxious child who saw herself as somewhat the victim of rather hostile forces in her world which she was unable to control. She perceived people in a distorted, rather frightening manner, and thus withdrew from close relationships. She had difficulty, I think, in acknowledging and accepting her own feelings, particularly her hostile and sexual ones, and dealt with them partially by projecting them on the outside world. There were indications that she had fantasies about eating in which one may be poisoned by food or where oral impregnation may take place through the ingestion of food. In the past she has been able to function with relatively good reality contact through the use of her various defenses. However, in the past year her life experience has put new stress on her. Her father has died and she has had to cope with the onset of puberty.

In the presence of these increased stresses she has shown increased withdrawal and a great deal of magical thinking. My feeling and the feeling of the department was that unless this child was given some sort of inten-

sive psychotherapeutic help even beyond the period of symptom relief, she might resolve her conflicts in terms of a schizophrenic adaptation.

Dr. Lourie:

To understand the development of this type of defensive symptom, as we might describe the severe anorexia, we have to look far back into an individual's thinking and experience. We have to consider the ordinary, everyday thinking that the little child goes through as it tries to interpret what goes on around it and what is happening to its body. If, in its attempt to find answers to these problems, the child is pushed aside, it may have to find it's own answers. In other words, the normal two to three year old may come up with some very weird, and to most of us pretty funny ideas of what is happening to the body. If these unusual ideas are not communicated and are not worked out they can remain encapsulated and may not bother the individual unless something comes up to call them forth. In the presence of some later stress, even in adult life, these ideas can take over. When this earlier kind of thinking comes to the fore it seems like a departure from reality. Probably in this case the early distorted idea of what happened to the body when food came into it, the way the physical sensations were interpreted by a young child took over, became again available to this girl, and then formed the basis for her change in eating patterns.

The idea of oral impregnation is not an unusual one in these cases of anorexia nervosa on the basis of the young child's idea of pregnancy. The patient thinks that she is pregnant, or maybe wants to avoid pregnancy by avoiding food. For example, I am reminded of one of my neighbor's youngsters who decided the other day that she was not going to visit Mrs. Smith next door anymore because Mrs. Smith eats babies. It turns out that Mrs. Smith is pregnant and this means that Mrs. Smith has a baby in her stomach. How did it get there? There was only one way the child could figure this out. She had been previously told that babies grow from a seed in the mother. How does the seed get there? She knows how things get into the stomach. If this is a lady who eats the kind of things that become babies the child believes she had better stay away from her. The child's thinking is in terms of cause and effect, and in terms of what she can understand.

When some emotional stress, such as a sexual problem, comes along and releases or calls up these encapsulated childish ideas, the individual must either rework these ideas and come up with better answers, or let these previously encapsulated ideas take over in such a way that they interfere with functioning in general.

In this case I think we have seen one of these childhood ideas about food coming out of its encapsulated form because of some traumatic

incident (possibly the death of the patient's father), and little by little, influencing her eating patterns. As this idea gets to be less and less compatible with what is going on around her, the rest of her thinking related to reality deteriorates in order to maintain the childhood idea. Then we see her turning in many different directions for ways to manipulate her feelings in thinking about people so that this idea will remain undisturbed. If this goes on long enough and becomes sufficiently widespread, she becomes more and more out of contact with reality and out of tune with what is going on around her. We then think of her as a very sick child, probably best described as schizophrenic.

This symptom of severe anorexia does not, per se, mean schizophrenia. It can be of a much less serious degree in terms of its over-all meaning to the individual. It need not occur exclusively in individuals having to protect themselves in as drastic a way as this child does, so that we may get a neurotic instead of a psychotic picture along with the anorexia. I do not think very many of us will forget that the trauma can be not only psychological but also organic, since our last case of classical anorexia nervosa was seen in a 9 year old child who died of a brain tumor.[13]

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PERFORATION OF THE COLON IN INFANCY

Byron D. Roseman, M.D. *

Perforation of the colon is not commonly encountered, particularly in the neonatal period. A small number of case reports have appeared in the recent literature. Some deal with meconium peritonitis and possible fibrocystic disease[1, 2], others with anomalous obstructions[3, 4]. Spontaneous or unexplained perforation without primary obstruction or other patent cause is quite unusual, however. Standard recently published such a case[5] and one is presented here.

CASE REPORT

S. A. M., an 11 day old white male infant, entered Children's Hospital because of abdominal distention and no stools for four to five days. He was the product of a second uneventful pregnancy in a mother who was delivered by uncomplicated low forceps technique. Birth weight was 8 pounds, 15 ounces, and examination at birth was not remarkable. He progressed normally, feeding on an evaporated milk formula for one week. Up to then, stools were said to have been yellow and pasty, numbering usually five daily. At about seven days of age they became drier and less frequent, and the most recent were blood streaked. Three to four days prior to entry they ceased to be passed entirely. No straining was noted. On the day of admission the infant regurgitated small amounts but there was no overt vomiting and he had continued to feed fully and well. Twelve to 18 hours before admission he passed flatus. Two enemata were given on the day of admission without result.

When examined, this fully developed and well nourished white male infant was crying vigorously and had a markedly distended abdomen. Pulse was 180, temperature 100°F., and weight 8 pounds, 9 ounces. Skin color and turgor were good. There was a small hemorrhagic area on the sclera of the right eye. The abdomen was markedly distended and moderately tense with a generally increased venous pattern. The percussion note was tympanitic outward to the flanks and no fluid wave was elicited. The bowel sounds were diminished in frequency and regularity, but normal in quality. There were no palpable masses or organs. The genitalia were normal male with both testes in the scrotum; there was no evidence of hernia. Rectal examination to 5 cm. revealed full patency, no mass, and no feces.

A small amount of yellow stool was passed when the admission temperature was taken, and in the few hours following admission the patient twice vomited moderate amounts of yellow and greenish material.

Six hours after admission x-ray examination of the abdomen in the supine and upright position revealed numerous loops of dilated small bowel and some fluid levels having the appearance of an organic obstruction in the region of the terminal ileum. Examination of the colon by means of a barium enema revealed the caliber of the large bowel to have been smaller than normal until barium reached the cecum where the bowel was noted to be markedly distended. Post-evacuation film revealed most of the barium to have been expelled. The previously noted distended cecum was still present. The nature of these changes was not known.

It is of interest that retrospective examination of the films showed the site of perforation (Fig. 1).

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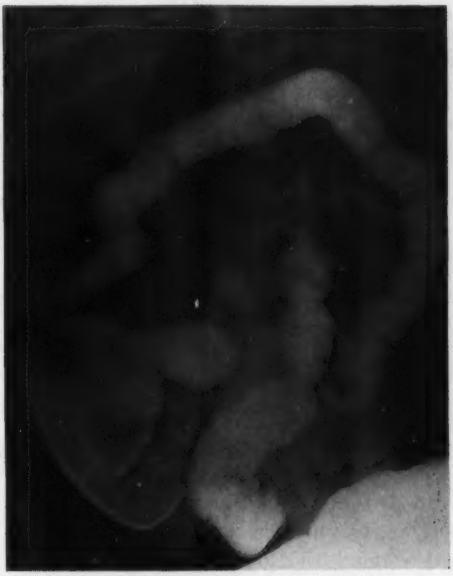

FIG. 1. Barium enema showing site of perforation (arrow)

Admission total white count is not available. Post-operative white blood cell counts averaged about 35,000 cells per cu. mm. Differential leukocyte count showed 5 per cent segmented neutrophiles, 14 per cent bands, 20 per cent young cells, and 12 per cent myelocytes, 1 per cent eosinophiles, 35 per cent mature lymphocytes, 1 per cent atypical lymphocytes, 7 per cent monocytes, and 5 per cent disintegrated cells. Hemoglobin was 14.1 gm. per 100 ml., and the hematocrit 47 per cent.

Ten hours after admission the patient was taken to the operating room for laparotomy with a preoperative diagnosis of intestinal obstruction at the terminal ileum of unknown nature.

When the abdomen was opened the peritoneum was thick and edematous; there was cloudy exudate throughout the peritoneal cavity. Distention of small bowel, cecum, and ascending colon extending to the hepatic flexure was noted. There was considerable inflammatory reaction along the right peritoneal gutter with free fecal matter in the peritoneal cavity about the hepatic flexure. Distal to the hepatic flexure the colon was constricted. Just proximal to the hepatic flexure the ascending colon was adherent to the liver, and as this was freed, an abscess cavity was opened, disclosing a perforation of the colon. Because of the above finding it was felt that the perforation had been present for several days. The cecum was so distended that tiny diastatic perforations were seen through the wall of the bowel under the serosal surface. Handling of the bowel caused these to open and fecal matter oozed into the peritoneal cavity. Surgery was completed with resection of a portion of the terminal ileum, and the cecum, appendix, ascending colon, and a portion of the transverse colon. A side-to-side ileo-transverse colostomy and Penrose drainage of the peritoneal cavity was performed.

At the conclusion of surgery the patient's condition was poor, with early shock

manifested by pallor and a pulse rate of 220 per minute. With blood, oxygen, and warmth, the infant improved, but on the first post-operative day it became necessary to institute gastric suction because of vomiting. Suction and oxygen were continued for five days. Penicillin and dihydrostreptomycin were given for seven days post-operatively. On the first day post-operatively his temperature rose to 102 degrees, and for the next four days to 100 degrees. Subsequently body temperature remained at a normal level. Oral feeding with a dilute formula and water was begun on the fourth day after surgery. Stools were passed daily from the day of surgery. Bowel sounds, however, were not definitely heard until the fourth post-operative day.

Despite some sloughing of the wound the patient progressed excellently and was discharged from the hospital on the twenty-first post-operative day. His discharge weight was 9 pounds.

Microscopic examination of the ascending colon at the site of perforation showed abscess formation of the mucosal surface with the underlying wall filled with acute purulent exudate. The acute inflammation extended throughout the entire thickness of the wall and adjacent to this was the perforation.

DISCUSSION

Compare the clinical picture in this case with the precise portrait drawn by Thelander[6]:

"An infant with perforation peritonitis present at birth or soon after, presents a fairly classic picture. The little patient looks sick. He is cyanotic, the respirations are rapid and grunting, the abdomen is distended and the abdominal wall, the flanks and the scrotum or vulva are usually edematous. Frequently brown induration of the edematous area which may resemble erysipelas is also present. Food is taken poorly or not at all. Vomiting is frequent and persistent. The vomitus contains bile and may contain blood. The stools are either absent or scant. Some mucus or blood may be passed. The temperature may be sub-normal, but varying degrees of fever have been reported. The blood count is of little or no value. The hemoglobin content may be very high, which probably indicates only dehydration. The leucocytes may or may not respond with a rise. In cases of intra-uterine perforation the abdomen has at times been so distended at birth that delivery was impossible until paracentesis of the abdomen has been performed".

Free fluid may also be present, and in the male a scrotal mass communicating with the peritoneal cavity which yields an impulse is presumptive evidence of free fluid[2].

In reviewing the medical literature from 1825 to 1939, Thelander[6] collected a series of 39 infantile gastrointestinal tract perforations including 25 of the large bowel. In this group, ruptures occurred twice as often in the large as in the small bowel, and in the entire series (large and small intestine) male patients predominated over female, three to one.

Review of the pertinent literature brings to light few similar cases. Those of Standard[5]. Thelander[6], and one of Franklin and Hosford[2] bear mention as relevant. However, perforation or rupture is a rare lesion

TABLE I

Etiologic Factors in Perforation of the Colon

I. Obstructive
- *Congenital*
  - Atresia[7]
  - Imperforate anus
  - Peritoneal bands
  - Malrotation[6]
  - Functional, Hirschsprung type
  - Duplication[5]
  - Bowel wall defects[6]
    - Muscular layer maldevelopment
    - Weakness at site of blood vessel penetration
    - Anomalous lymphoid tissue predisposing to diverticuli
- *Acquired*
  - Hernia, including umbilical[8]
  - Volvulus
  - Intussusception
  - Adhesions
  - Neoplasm
    - Malignant
    - Benign
  - Angulation, kinking associated with early stages of diverticulosis[9]
  - Endometriosis[10]

II. Mechanical & Traumatic
- Foreign body[11, 12]
- Fecalith
- Instrumentation[13]
- Intraluminal gas explosion
- Compressed air, extrinsic[14]
- Birth trauma*[15]
- Exertion[9, 16]
- Non-penetrating trauma to abdomen[17]

III. Infectious
- *Primary*
  - Local inflammation, including diverticulitis[6]
  - Typhoid
  - Bacillary dysentery
  - Amoebiasis[18]
  - Tuberculosis
  - Helminth infestation
- *Secondary*
  - Pancreatitis
  - General sepsis[6]

IV. Metabolic
- Fibrocystic disease
  - Meconium ileus
- Steroid therapy
  - Perforating diverticulitis[19]

V. Neoplastic
- Primary malignant perforation

VI. Vascular
- Vascular occlusion
- "Primary vascular insufficiency"[5]

VII. Idiopathic
- Unexplained perforation
- Ulcerative colitis
- Simple colonic ulcer[20]

* Birth trauma as a cause of perforation has logically been questioned by a number of authors[4, 5, 6].

in both pediatric and adult groups. The causes are legion. In the present case the pathogenesis is in the realm of conjecture. Table I is a classification of the known causes of perforated colon. It includes a number of factors clearly inapplicable to the pediatric, and particularly the newborn age group.

The operative findings in the present case, in addition to the primary

site of pathology included spreading, separation, and diastasis of the fibers of the musculature of the cecum. Saeltzer and Rhodes[21] discuss this phenomenon. In the presence of a competent ileo-cecal valve with large bowel obstruction distally, diastatic perforation tends to occur in the cecum, because, of the entire bowel:

1. The cecal wall is thinnest and experimentally most subject to rupture secondary to increased intraluminal pressure.

2. The cecal portion is least capable of hypertrophy in response to distal obstruction.

3. Cecal diameter is greater than the diameter of other parts of the colon.

Aschutz[22] in an ingeniously simple experiment showed how the larger diameter of the cecum places that viscus in greater jeopardy in the presence of obstruction distal to it. He connected rubber balloons of unequal size to either end of a T-tube. It was found that if water were forced into the leg of the T, the larger balloon consistently ruptured first. Saeltzer and Rhodes[21] discuss the mathematics involved in this phenomenon.

Recovery from perforated colon in infancy has in the past not been frequent. More recent writing on the topic, however, including the present case, would seem to lend a more optimistic note.

In order to promote recovery from perforation of the colon the following are essential:

1. Consider colonic perforation as a part of the differential diagnosis of intestinal obstruction with abdominal distention.

2. Make full use of the roentgenogram. X-ray examination in the first 24 hours of life may show no air in the gut; during this period, without at least one grossly dilated loop, intestinal obstruction cannot be diagnosed by this means. After 24 hours of age, however, unless air is present in the rectum, obstruction may be assumed[2]. Pneumoperitoneum with or without hydroperitoneum may be manifest and is an indication for immediate surgery; in their presence administration of barium is unwarranted and potentially harmful. Latent or walled-off perforations may have neither of these features, however, and the use of a contrast medium may be necessary.

3. Delay in instituting surgical investigation of the lesion cannot be condoned beyond time necessary to prepare the patient for anesthesia with blood, fluids, and electrolytes. Local anesthesia may be necessary in the individual case.

4. Severe abdominal distention causing respiratory embarrassment should be relieved by paracentesis of the abdomen prior to surgery[5].

Discussion of surgical techniques involved is not within the scope of this paper and is readily available in standard surgical references.

SUMMARY AND CONCLUSIONS

Perforation of the colon in the neonatal period is an unusual, infrequent occurrence. The case of an 11 day old infant presenting with abdominal distention and obstipation is discussed. The patient underwent surgery which disclosed a walled-off perforation of the hepatic flexure and secondary diastatic perforations of the cecum. The cause was undetermined. Recovery was uneventful.

Progressive abdominal distention, vomiting, and pneumoperitoneum (by x-ray) are the hallmarks of colonic perforation.

The cecum, because of its relatively larger volume and thinner wall is more subject to diastatic perforation than are other parts of the colon.

An etiologic classification of perforation of the colon would include obstructive, mechanical, traumatic, infectious, metabolic, neoplastic, vascular, and idiopathic causes.

Essentials in the reduction of mortality resulting from this lesion are consideration of the diagnosis, judicious use of the roentgenogram, surgery as soon as feasible after proper preparation, and abdominal paracentesis to relieve respiratory embarrassment.

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ATRESIA OF THE COLON

George R. Dalton, M.D. *

Atresia of the intestinal tract is variously estimated as occurring once in every 1,500 to 20,000 live births. The following is a case of atresia of the colon of an uncommon type in which there was discontinuity of the affected segments, and which occurred in an unusual location.

CASE REPORT

M. S., a colored female infant was born at a local hospital on August 31, 1956 after an uncomplicated term pregnancy, the mother's tenth. The mother and other siblings were all living and well and there was no history of previous abnormality occurring in the family.

Delivery was normal, and the infant's respirations were established spontaneously at birth. At that time she was described as a normal, vigorous negro girl, weighing 6 pounds and 14 ounces. When the infant was first nursed, at 12 hours of age, slight abdominal distention was noted. Subsequently, she passed a small amount of meconium. When she was 18 hours old, rectal examination by a pediatric resident revealed an obstruction 2 cm. above the anal opening, which yielded readily to pressure and permitted full entrance of the examining finger. The stomach was lavaged with normal saline. The infant continued breast feedings and at 36 hours of age was given supplementary feedings of 5 per cent glucose in distilled water. Another small meconium stool was reported.

At 42 hours of age the infant was again seen by a pediatric resident because of an

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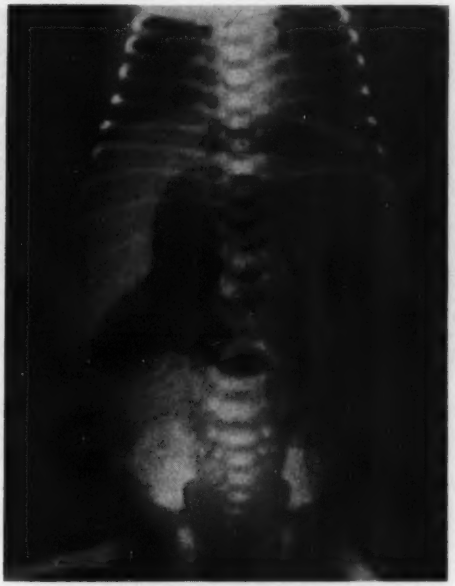

FIG. 1. Plain film of the abdomen in the upright position shows numerous loops of distended small bowel. No definite large bowel is visualized. There is an absence of gas in the lower half of the abdomen. These findings suggest a complete, organic obstruction in the lower intestinal tract.

episode of bile stained emesis. At that time the abdomen was described as distended but not tense. No masses were palpated, but upon pressure over the suprapubic area, urine was expressed. The bowel sounds were high pitched in character but the peristaltic rhythm was normal. The rectum was then irrigated with normal saline under gentle pressure and a 7.5 cm. mass of greenish white meconium of narrow calibre was removed. A Farber test unfortunately was not performed. Rectal examination following irrigation again revealed the sensation of obstruction at 2 cm. which yielded easily to pressure. There was, however, felt to be an obstruction 6 cm. above the anal orifice. A catheter was passed into the bladder and 5 ml. of grossly normal urine was removed. The infant was transferred to Children's Hospital for further diagnostic studies and treatment. The diagnostic impression was obstruction of the large bowel.

When the infant was 48 hours old x-rays of the abdomen in the upright and supine positions revealed the small intestine to be markedly dilated. The large bowel was poorly visualized and no fluid levels were seen (Fig. 1). A barium enema was immediately performed with the enema can two feet above the infant's rectum. The barium reached the proximal end of the sigmoid colon (Fig. 2), but because it could not go further an immediate operation was planned.

Following aspiration and decompression of her stomach, the infant was taken to the operating room, where a left rectus incision was made. When the peritoneum was

FIG. 2. Barium enema demonstrates a normal rectum. The sigmoid colon is narrowed and it was not possible to propel the barium beyond the visualized point. These findings suggest an organic obstruction in the midportion of the sigmoid colon.

opened dilated loops of small bowel were observed. The proximal part of the transverse colon ended blindly in the left transverse area; the distal transverse and descending colon, and most of the sigmoid colon were absent. A loop of the left transverse colon was mobilized and exteriorized, and the bowel decompressed by needle puncture. Thirty-six hours later the colostomy was opened by cautery.

Two and one-half months later the infant was discharged to a convalescent home weighing only 7 pounds, 6 ounces, following a very stormy postoperative course characterized by constant diarrhea, abdominal distention, electrolyte imbalance, negative nitrogen balance despite caloric intake of up to 100 calories per pound of body weight, and periods of urinary retention.

DISCUSSION

Gross states that atresia of the intestinal tract occurs once in every 20,000 live births. Evans, after a review of the world literature, stated that a more accurate figure would be one in every 1,500 live births. If these statistics are correct there must be several thousand infants born with atresia of the intestinal tract in the United States each year.

Most of the embryological development related to atresia of the gastrointestinal tract takes place between the fifth and twelfth weeks of fetal life. Before the fifth week, the fetal intestine is tubular in shape and has a well defined lumen lined with epithelium. At about the fifth week, intestinal epithelium begins to proliferate. The lumen of the intestine from the pylorus to the ileo-cecal valve becomes obliterated by the epithelial

growth, and the tubular intestine is temporarily transferred into a solid structure. Vacuolization then takes place and when complete, an intestinal lumen is once more present. It is definitely known that such a solid stage normally exists in the human small intestine but there is some question concerning the presence, extent, and duration of the solid stage in the colon.

Stenosis is believed to occur if the process of vacuolization does not result in a normal intestinal diameter, while atresia is believed to occur if the vacuolization process fails at any point to reestablish a lumen. By such definition it would appear incorrect to use the term partial and complete atresia. Also, if during its solid stage a portion of the intestine becomes pinched off, an atresia with discontinuity of the proximal and distal intestinal segments results. The present case is an example of this. A third type of atresia presents as a thin fibrous cord of variable length connecting the proximal and distal intestinal limbs. Whichever of the three types of atresia is present has little bearing on the presenting signs and symptoms of atresia, but is understandably of extreme importance to the surgeon. Regardless of location the intestine distal to the obstruction is very small, contains no gas and usually has within its lumen only small amounts of mucus and cellular detritus which have been cast off from the lower intestinal mucosa. This collapsed small intestine measures no more than 4–6 mm. in diameter and a collapsed colon is not much larger. In the case presented the apparent obstruction 2 cm. from the anal orifice was most probably the collapsed rectum.

The diagnosis of intestinal atresia should be made in the first 48 hours of life. Examination at birth reveals a normal anal canal. Although no true meconium will be passed, a small amount of light-colored mucoid material may be noted. In the present case, however "meconium" was twice reported in the nursing notes, and the plug removed following saline irrigation of the rectum was fairly large and a light green color suggestive of bile staining. It was not the whitish mucoid material so often described in the literature as characteristic. One wonders whether this plug would have tested positively for the presence of bile. Bile stained stool may be found in cases of biliary atresia and the mechanism is said to be secretion of this substance at the colonic level. The important practical point to be made, however, is that a diagnosis of intestinal obstruction should not be dismissed because "meconium stools" are passed by the infant. These "meconium stools" may be examined for vernix cells and if none are found it may be assumed that there is a complete obstruction in some portion of the intestinal tract.

In the case of colonic atresia the abdomen becomes distended, but extreme distention is uncommon before 36 hours of age. An intestinal

pattern with peristaltic waves may be seen over the abdomen. Vomiting is not an early symptom with large bowel obstruction and is usually not seen before the second or third day of life. Dehydration from vomiting and from loss of fluids into the distended bowel is rapid in infants and leads to temperature elevation and toxicity. However, in the present case the infant's abdomen was distended but not tense and demonstrated no peristaltic waves (although the bowel sounds were high pitched in character). The infant vomited but once in 48 hours and did not appear very toxic.

X-ray examination of the abdomen is of great assistance in visualizing the level of obstruction. It is rarely necessary to use contrast medium and barium should never be given by mouth. Barium enema is occasionally helpful but the danger of perforation of the blind or obstructed segment must be kept in mind. X-rays of the abdomen in the supine and upright position will usually provide the desired information. In colonic obstruction the gas pattern usually outlines the colon quite well up to the point of obstruction, and the upright film usually reveals fluid levels. In the present case visualization of the colon was uncertain and fluid levels were not seen. The radiographic picture was more characteristic of that described with inspissated meconium.

Gross has reported 140 cases of atresia of the intestine and colon in a 12 year series of which 32 occurred in the duodenum, 19 in the jejunum, 72 in the ileum, 2 at the ileo-cecal valve, 6 in the colon, and 9 in multiple sites. He states that the level of an atresia has little bearing on the prognosis. His series during the years 1940 to 1952 shows approximately the same proportion of survivals and fatalities regardless of the level of alimentary tract atresia. However, in the surviving cases there does seem to be an important difference in the postoperative course which bears some relationship to the level at which the atresia occurred. Those who had an atresia in the duodenum or jejunum usually recovered from surgery quite promptly and had a smooth postoperative course thereafter. In contrast, a few whose atresia was in the lower intestinal tract had a long convalescence characterized by poor nutrition, feeding difficulties and bouts of partial obstruction which were almost entirely related to the presence of intestinal adhesions. These complications have not been appreciably reduced by antibiotics or the aseptic operations offered by the Mikulicz method of anastamosis. Peritoneal adhesions account for three-fourths of the operative failures in Gross' series. Of 17 infants with atresia at all levels operated on in 1952, Gross' survival rate was 70 per cent.

SUMMARY

Congenital atresia of the small intestine and colon is an uncommon malformation. The exact incidence is disputed. The presenting signs and

symptoms vary depending upon the level of obstruction. Without surgical intervention death ensues usually within a week. Surgical intervention is followed by a high mortality, particularly due to recurrent obstruction, but the survival rate is increasing. Prompt recognition of the obstruction and surgery as soon as fluid and electrolyte balance is attained, give the only chance of survival.

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